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PARALYSIS OF THE LATERAL ADDUCTOR MUSCLE OF THE LARYNX,

WITH UNIQUE CASE.*

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Paralysis of the adductor muscles of the larynx, as is well known, is most infrequent; it is the *abductor* muscle or nerve which is affected in the vast majority of cases. Semon says in this connection: "Not only have I never seen such a case, but in the whole range of laryngeal literature which is known to me I have been unable to find a *single case* in which primary organic † disease of the brain or nerve trunks was proved by clinical observation or the result of post-mortem examination to have been the cause of isolated paralysis of the adductors." Morell Mackenzie gives five cases in all of paralysis of one lateral adductor; in one case only was there a post-mortem made, and in this, though the muscle itself was atrophied, there was no dis-

* Read before the Clinical Society of Baltimore, January 16, 1887.

† The word organic is used because it is well known that the functional neuroses show a strange predilection for affecting the adductor muscles.

ease of the nerve trunk. He has met with this form of paralysis after small-pox and from syphilis. Cohen says it may result from organic cerebral disease, from phthisis, from metallic poisoning, from injury to or pressure upon the nerve, etc.

However this may be, the affection is very rare, and, although in the following case I have no positive proof of disease of the brain or nerve trunks, I am satisfied that the lesion was a paralysis of the *left lateral adductor*. Owing to the importance of the observation, I asked my friends Dr. H. Clinton McSherry and Dr. Samuel Johnston to examine the case. They did so upon separate occasions, and both confirmed my diagnosis. Briefly the case is as follows:

E. M., a farmer, aged fifty-three, consulted me in August, 1886, for partial loss of voice, which had come on somewhat suddenly eight months before, and which had become gradually worse. He was and had always been a strong, healthy man, accustomed to an out-door life. He had never been ill in his life. There was absolutely no history of either rheumatism or syphilis. Some months before I saw him he had suffered, however, with a very severe pain located behind the right ear, following which came considerable deafness in the left ear, with loss of voice. On laryngoscopic examination, the left cord was seen in extreme abduction, immobile during respiration and upon phonation; and, although the right cord was well adducted beyond the middle line, there was still a considerable aperture between the vocal bands. The arytenoids were healthy, and this fact was specially noted, for it is well known that symptoms of a paralytic character are produced by destruction or impairment of one of the crico-arytenoid joints from ossification or other morbid change; and in these cases there is generally some abnormal appearance, such as enlargement or swelling about the base of the arytenoid cartilage. My experience, too, has been that in the latter class of cases there

is gradually more or less movement of the cord on the affected side. In the case in question there was absolutely no movement. Again, ankylosis of the arytenoid results from a perichondritis following typhoid fever, syphilis, rheumatism, or gout, and there was no such history in this patient. Finally, the attack of severe pain behind the ear, with the deafness and loss of voice which followed, pointed to a brain lesion as the cause of the trouble.

Treatment was of no avail, and at last accounts there had been no change in the patient's condition.

The uniqueness of this case naturally suggests the question, Why in both central (brain) and in peripheral (nerve trunk) lesion are the *abductors* almost invariably affected? Or, to put it more broadly, why, in such cases, do we find the cord fixed in the middle line (in the phonatory position)? There are several explanations of this fact:

1. Semon holds that there is a natural "*proclivity*" of the abductor fibers to disease, and he looks on all cases where the cord remains fixed in the middle line as examples of true paralysis of the posterior crico-arytenoid muscle.

2. Krause, on the other hand, maintains that, "in analogy with neurotic manifestations in other portions of the body, organic irritation of the recurrent laryngeal nerve produces spasm of all the muscles supplied by its fibers, both those which preside over the patency of the glottis for the respiratory purposes, and those which preside over the approximation of its edges for purposes of phonation, cough, or expulsion of foreign material; but that, inasmuch as the number and mass of muscles and nerve fibers predominate in the domain for closure, the equilibrium maintained by the respiratory center under normal conditions is overpowered, and the spasm therefore manifests itself in the closing phase only, despite the co-existent spasm of the dilator muscle. The opinion held, then, is that the phenomenon of

permanent fixation is due to the overpowering contracture or final spasm of the contracting muscles of the larynx, and not to a paralysis of the dilating one. The atrophy of the dilating muscle is attributed to its mechanical, not to its paralytic immobility."

Such are the two chief theories with regard to this question of laryngeal paralysis. I do not propose in this paper to give a list of authorities who agree with the one or the other, but desire here to define my own position in the matter, and to offer an explanation of the phenomena of this so-called abductor paralysis. In a paper read before the American Laryngological Association in 1886, I stated that my experiments gave no confirmation of Semon's assertion. On further consideration, I find this statement too broad. Whereas I was at first inclined to deny Semon's statement (on scientific grounds), I now go almost as far as he does—*i. e.*, I am fully convinced that *the abductor fibers are much more irritable and have much less power of resistance than the adductor fibers, and that they die sooner;* though I can not say that I have as yet any experimental (histological) proof that they degenerate more rapidly, after chemical, mechanical, or electrical stimulation, than the adductor fibers.

In the experiments related in that paper I also found that the abductor muscles responded to a much weaker stimulus than the adductors did, and that this result invariably followed—whether the dog was slightly, deeply, or thoroughly narcotized, whether the animal was eupnœic or apnœic—when the medulla had been destroyed and after local death had taken place. This important fact (that the adductor muscle does respond to a slighter stimulus) has been confirmed by Semon and Horsley. In a paper "On an Apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles," in the "British Medi-

cal Journal" for August 28, 1886, page 405, they say: "With weak stimuli, other things being equal, *abduction* of the vocal bands generally occurs, as has been recently shown by F. Donaldson, Jr. We have repeated in a few instances Dr. Donaldson's experiments, and have, on the whole, obtained similar results." My observation has, I understand, also been confirmed by a distinguished American physiologist, *provided the animal is slightly under ether.*

Again, I found (and this, too, has been confirmed by Semon and Horsley) that, when after death the recurrent laryngeal nerve was stimulated, *abduction* disappeared long before *adduction*, which would certainly show that the *abductors die* or become exhausted first.

In view of the foregoing facts, I am forced to agree with Semon that those cases where the vocal band is found fixed in phonatory position are true paralysis of the *abductor* muscle, and not spasm of the *adductor* muscles. Moreover, the constant implication of the abductor muscle may be explained on the ground of the greater irritability of the abductor muscle or nerve fibers. *For in cases of unilateral or bilateral lesion of the cords from an aneurysm or tumor the constant pressure exerted by either upon the nerve acts as a mechanical stimulus to it, and the more irritable abductors are, therefore, the first to show the result of this constant stimulation in their loss of function.*

Another factor in the fixation of the vocal band in the phonatory position in the majority of cases (as shown by Dr. Gowers) may be the mechanical advantage at which the chief adductor acts, as compared with the chief abductor, which gives greater power to the former, since it passes nearly at right angles (while the abductor passes at a very acute angle) to their identical insertion into the muscular process at the outer angle at the base of the arytenoid car-

tilage. Any loss of power would, therefore, affect the abductor muscle most.

Finally, it is but right to confess that my experiments tend to confirm Semon's conclusion.

I hope in a later paper to develop further this theory of a greater irritability of the abductor muscle, which I think goes a great way toward explaining its so frequent paralysis.